# Selective Trans-Splenic Decompression of Gastroesophageal Varices by Distal Splenorenal Shunt

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Life-threatening hemorrhage is frequently an indication for surgical therapy. In patients with cirrhosis of the liver and esophageal varices, variceal bleeding is the leading cause of death and often contributes to mortality from other sources, i.e., hepatic failure.[6] The efficiency of the portacaval shunt in controlling hemorrhage has been well documented.[7] With proper selection of patients, operative mortality can be lowered to acceptable levels. In considering the surgical treatment of bleeding due to portal hypertension, several questions have given cause for concern.

*1. What are the long-term results of portacaval shunts?* Recent reports by Callow *et al.*,[2] Conn and Lindenmuth,[3] and Jackson *et al.*[12] have analyzed critically the results of shunt therapy in well-controlled, randomized studies. In spite of a low operating mortality, survival figures have not demonstrated the superiority of the surgical group (Fig. 1, 2). An analysis of the cause of death reveals substantial differences between the two modes of therapy (Fig. 3). The Boston and Hartford series were utilized in this study because the patient population is more representative and the judgments more consistent than in a large, national study. Following portacaval shunt, the death rate from hepatic failure is greatly increased and completely offsets the undeniable protection afforded against fatal hemorrhage. A logical explanation would be that the operative mortality accounted for the increased number of deaths from hepatic failure. This is not the case, however, as there was only

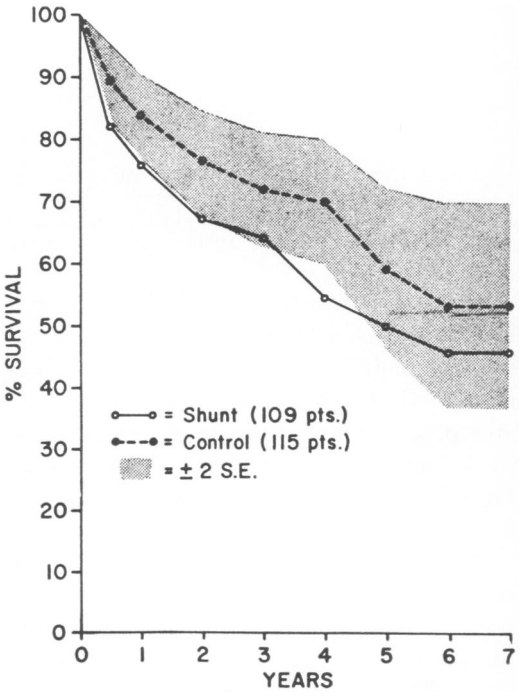


Fig. 1. Comparative survival in randomized patients undergoing prophylactic portacaval shunt. From Grace *et al.*[7] Reprinted by permission of *Gastroenterology*.

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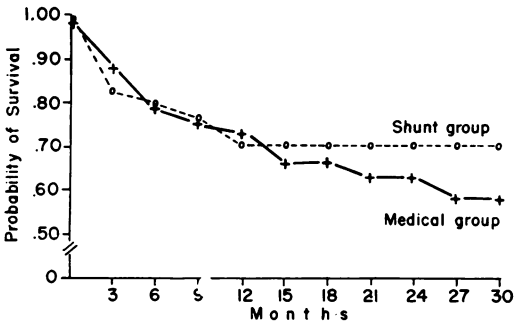


FIG. 2. Comparative survival in randomized patients undergoing therapeutic portacaval shunt. From Jackson *et al.*[12] Reprinted by permission of *Arch. Surg.*

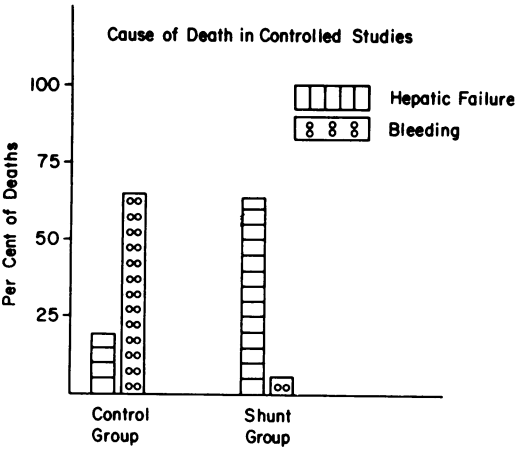


FIG. 3. Comparison of cause of death in shunted and non-shunted patients in controlled series.[2, 3] All patients reported are included (randomized, refusal, exclusion and therapeutic shunt groups).

one actual operative death in the combined series; a few patients were never well enough to leave the hospital. These data indicate that portacaval shunt initiates or accelerates hepatic deterioration in some patients and the anticipated death rate from hepatic failure is substantially increased.

2. *Would any major operative procedure accelerate hepatic failure comparably?* Although there is no definitive answer to this question, the available evidence indicates that other than the acute changes incident to operation little or no further hepatic deterioration results from non-shunting procedures. Womack and his colleagues have used non-shunting procedures routinely and their most recent experience has been reported by Johnson, Dart, Peters and MacFie.[13] Although the operative mortality was high (emergency procedures were included) delayed hepatic death was rarely seen and there was favorable long-term survival (Fig. 4).

In an important study, Hassab has reported the results of non-shunting procedures for bleeding esophageal varices in bilharzial cirrhosis.[9] Such patients might appear to be excellent candidates for portacaval shunt as they demonstrate large varices and marked portal hypertension while maintaining good hepatocellular function. However, dissatisfaction with late results has led him to virtually abandon portacaval shunts. In 355 patients with gastric devascularization and splenectomy there was only a 2.6% death rate from hepatic failure (all immediately postoperative), one late case of jaundice and one patient with encephalopathy. In "comparable patients" treated by portacaval shunting, "jaundice and liver failure occur with increasing frequency as time passes." This

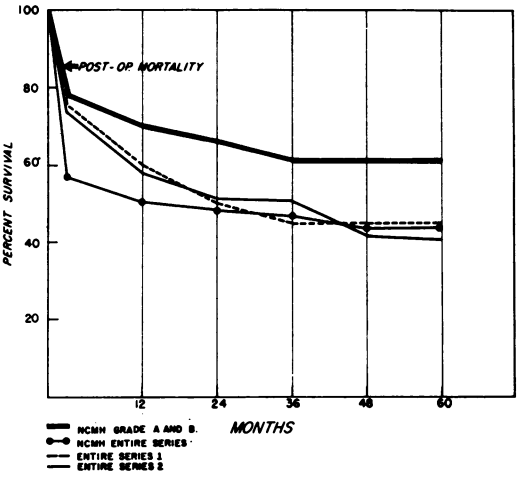


FIG. 4. Series 1 and 2 depict survival following portacaval shunt as compared to the N.C.M.H. non-shunt series. The top curve is corrected by elimination of Grade C patients to more nearly match the degree of illness with the portacaval shunt patients. From Johnson *et al.*[13] Reprinted by permission of *Ann. Surg.*

has been confirmed by other experienced surgeons.[9]

The detrimental effect of sudden, total, portal deprivation with splanchnic venous decompression is well documented.[18] It has long been known that normal dogs tolerate portacaval shunts poorly and usually die in hepatic failure within a few months. A similar syndrome has been observed in our laboratory with primates (spider monkey).[5] The few cases of portacaval shunt with normal liver and unobstructed portal vein in man have developed a similar syndrome.[11]

An excellent study by Mikkelsen of noncirrhotic portal hypertension contains important data that have received scant attention.[14] Among the patients reported, 17 had a patent portal vein, no evidence of liver disease grossly, and excellent liver function. In this group, usually called "idiopathic portal hypertension," there was nothing to suggest liver decompensation prior to operation. Following portacaval shunt, 10 developed signs of encephalopathy or liver failure and five died in hepatic coma. These complications are rare following non-shunting procedures.

3. *Should "non-shunting procedures" be used routinely?* Routine use of non-shunting procedures has been quite successful in bilharzial cirrhosis and in selected cases of Laennec's cirrhosis.[20] General application to a group of cirrhotics, however, leaves much to be desired. The chief problem appears to be bleeding, both in the immediate and late postoperative period. In the study by Johnson *et al.*,[13] there were 22 operative deaths (43%) of which six were due to continued bleeding. Of the surviving patients, 45% had recurrent bleeding, 31% required reoperation and 14% died as a result of this complication. Encephalopathy was not encountered in the surviving patients.

The major advantages of this procedure, in our opinion, are the maintenance of an elevated intestinal venous pressure and preservation of hepatopetal portal flow. However, this is offset, at least in part, by the persistent danger of gastroesophageal hemorrhage.

Despite the superior results of non-shunting procedures in stabilizing hepatic function and avoiding encephalopathy, it must be remembered that a good functional result from portacaval shunt is seen in perhaps 40–50% of cirrhotics and affords maximal protection from bleeding. This figure is much higher in patients with an essentially normal liver and extrahepatic thrombosis of the portal vein, as the accommodation to loss of portal flow has already been made. The mechanism of adjustment to decreased portal venous flow is not clear. Deprivation of hepatopetal portal flow is tolerated much better if due to extrahepatic portal vein obstruction rather than to that produced by portacaval shunt.[1]

4. *Should splanchnic hypertension be completely decompressed?* Excepting the hepatic sinusoids, patent portacaval anastomosis abolishes the hypertensive state in the portal venous system and in the splanchnic capillary bed.[25, 26] Following a side-to-side portacaval shunt the wedged hepatic vein pressure may decrease but does not become normal. After an end-to-side anastomosis this pressure sometimes increases. Nevertheless, control of ascites is one advantage of the shunt procedure.[22] Two factors dull the lustre of such an achievement. First, there is the total loss of portal flow to the liver which follows both the end-to-side and side-to-side portacaval shunt.[24, 26] Second, the control of ascites by nonoperative means has improved remarkably and only rarely does one see truly "resistant ascites." Hypersplenism, a third major complication of portal hypertension is seldom completely relieved by return of portal pressure to normal. Only rarely is the condition severe enough to create a problem in management and

splenic artery ligation is a simple means of achieving a satisfactory remission.

Numerous studies have shown that a well-compensated stable cirrhotic patient, without esophageal varices, and with no further liver injury, may lead an essentially normal life. Recently, Price *et al.*[17] have shown the increased rate of absorption of ammonia and other substances following shunt decompression of portal hypertension. This may partially account for the relatively low incidence of portal systemic encephalopathy seen in non-shunted patients with cirrhosis.

It appears that the advantage of both the shunting and non-shunting operations can be achieved while avoiding the disadvantages of each. Ideally, an operation should allow continued perfusion of hepatic parenchyma by portal flow from the intestine and yet decompress the venous system in the gastroesophageal area. Gastrosplenic isolation with distal splenorenal shunt seems to meet these requirements.

### Experimental Demonstration of Trans-Splenic Gastric Drainage

Greyhounds of average weight 25 Kg. each were anesthetized with pentobarbital administered intravenously (35 mg./Kg.). Through a midline laparotomy incision the gastrosplenic vein was isolated and divided just proximal to its junction with the superior mesenteric vein. An anastomosis was constructed between the gastrosplenic vein and the inferior venacava. In four dogs, a polyethylene shunt was interposed between the femoral artery and the severed distal end of the gastric vein. Upon release of occlusion, pulsations were noted in veins which usually drain the cardia and fundus of the canine stomach.

Systemic arterial pressure was recorded continuously from a cannula through a Statham transducer on a GME direct writing recorder. Each animal received lactated Ringer's solution at the rate of 40–50 ml./Kg./hr.

Splenic blood flow was estimated by Xenon$^{133}$ clearance.[29] While the details are described elsewhere, a brief summary of the technics are presented to make the results intelligible. Radioactive gas or liquid was injected either directly into the tissue or into the splenic artery. Care was taken to insure constant geometric relationship between the sodium iodide crystal and the spleen. The output of the photomultipler tube was amplified for recording on a direct writer (Texas Instruments). The disappearance of peak radioactivity in the spleen was recorded and blood flow /100 Gm. splenic tissue/minute was calculated from the recorded disappearance curve (average 22–28).

In two dogs, a gastrosplenic–inferior vena cava anastomosis was performed under sterile conditions and the animals maintained for three months. Prior to sacrifice splenoportography was performed.

Splenic blood flow as estimated by Xenon$^{133}$ clearance in two dogs with gastrosplenic–inferior vena caval anastomoses was similar to the intact dog (22 and 25 ml. /100 Gm. spleen/min.) during occlusion of the femoral arterial-gastric venous shunt. Injection of the radioactive material into the open arterio-venous shunt was followed by detection of the radioactivity over the spleen with recorded activity curves similar to that found after injection of the Xenon into the splenic artery in the intact dog. However, splenic blood flow was reduced and the mechanism for this phenomenon is by no means clear. It is possible that reflex spasm of the splenic artery was initiated by hypertension in the short gastric and splenogastroepiploic veins. Further studies should provide additional insights into this fascinating problem.

In the first two dogs used for these acute studies, the detection of the blood contributed to the spleen by the femoral arterial-gastric venous shunt was attempted by estimating oxygen saturation in the splenic venous blood during both occlusion and

release of the arteriovenous shunt. No significant differences were noted and this was attributed to the normal high oxygen saturation of splenic venous blood in this species (88–92%). In addition, the reduction in splenic blood flow as discovered by the later Xenon clearance studies probably mitigated against finding an increase in the venous oxygen saturation.

In the two dogs allowed to survive for 3 months, splenoportograms have demonstrated patent anastomoses. Histological examination of the spleen has revealed no abnormalities.

The results of these studies have provided support for the concept that the spleen may serve as an outflow tract for the cardiac area of the stomach under the condition of venous hypertension. On the one hand, diversion of splenic venous flow into the systemic venous system is a simple technic and more important, the anastomosis remained widely patent at least for three months in the dog. This venous shunt in no way effects splenic blood flow as measured by $Xenon^{133}$ clearance nor is it associated with histologic changes in the spleen of chronic survivors.

Further, it is clearly demonstrated that a gross arterio-venous fistula involving the major gastric venous drainage in dogs results in some reversal of flow through the short gastric circulation into the spleen. The detection of $Xenon^{133}$ in the spleen following injection of the material in the femoral artery side of a femoral arterial-gastric venous shunt supports no other conclusion since $Xenon^{133}$ is not recirculated.

## Methods and Material

All patients were assessed by clinical evaluation based on careful history and physical examination, laboratory determinations of hepatic function and the appraisal of hemodynamic alterations. The technics used to evaluate hepatic and splanchnic vascular physiology included estimation of total hepatic blood flow, liver scan, hepatic vein catheterization, splenoportography or indirect portography and visceral angiography.[24]

Total hepatic blood flow was estimated (EHBF) using radioactive colloidal gold. The normal K value in this laboratory is $0.29 \pm 0.05$. In patients in whom liver blood flow is slightly reduced the value is $0.23 \pm 0.07$. With moderate reduction in flow K is $0.19 \pm 0.05$ and with severe reduction it is $0.16 \pm 0.05$. However, to interpret individual K values the amount of extrahepatic gold uptake must be estimated from the liver scan and the shape of the disappearance curve of $Au^{198}$ must be evaluated.

By hepatic vein catheterization it was possible to determine the degree of portal hypertension and to gain indirect information concerning the amount of hepatopetal portal blood flow. With the catheter wedged in an hepatic vein the pressure is recorded (WHV). The catheter is then moved into the free hepatic vein and the pressure is again recorded (FHV). By subtracting the latter from the former the corrected sinusoidal pressure (CSP) is obtained. A pressure between 6 and 14 mm. Hg signifies mild portal hypertension. Moderate hypertension exists when the pressure is between 15 and 20 mm. Hg. Above 20 mm. Hg the hypertension is severe. With the catheter in the wedged position radiopaque material is injected. Each venogram can be placed in one of four categories. In Category 1 there is excellent filling of the sinusoidal bed but no filling of the portal system. This is characteristic of those patients in whom portal flow to the liver is normal or only mildly restricted. In Category 4 there is hepatofugal flow through the portal vein and into the splanchnic circulation. Categories 2 and 3 are intermediate.

Splenoportography is usually the single best source for estimating portal hemody-

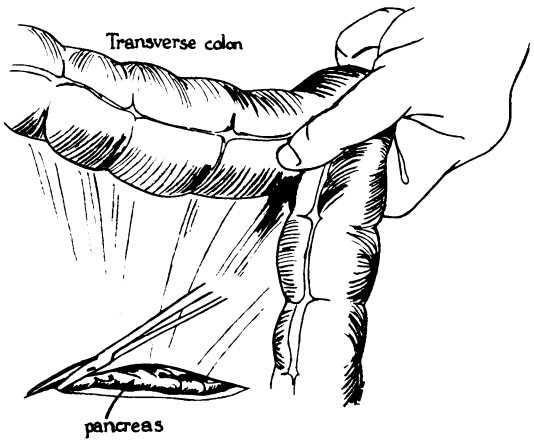


FIG. 5. With the transverse colon retracted upward and in a cephalad direction, the transverse mesocolon is stretched. An incision in the peritoneum at the root of the transverse mesocolon to the left of the superior mesenteric vessels exposes the inferior border of the body of the pancreas.

namics. Stage 1 represents normal or only slightly restricted hepatopetal portal flow. In Stage 2 portal flow to the liver is moderately restricted and in Stage 3 it is severely restricted. Total lack of opacification of the portal vein is the criterion for Stage 4. At times indirect portography is used in lieu of splenoportography. Staging is similar to that of splenoportography.

In presinusoidal hypertension and when the EHBF is relatively high and portal hepatic venous perfusion is decreased the hepatic artery may appear unduly large, perhaps because of compensatory hypertrophy. In these situations there is a prominent liver blush on the late films of the hepatic arteriogram.

Six patients who had bled from esophageal varices were studied. Liver function was relatively good in all and hepatic portal perfusion was good. Because of the morbidity associated with sudden decrease in portal blood flow to the liver four patients had a distal splenorenal shunt with gastrosplenic isolation and two had the stomach devascularized but the spleen was left in place.

## Technic of Distal Splenorenal Shunt and Gastrosplenic Isolation

A long midline incision is made. The transverse colon is lifted upward and in a cephalad direction. A transverse incision is made at the root of the transverse mesocolon to the left of the mesenteric vessels (Fig. 5). The inferior border of the pancreas is recognized readily and rotated gently in a cephalad direction. With careful dissection in the retroperitoneal tissue behind the pancreas and near its superior border, a short segment of splenic vein is identified close to its junction with the inferior mesenteric vein. Small venous tributaries from the pancreas are dissected meticulously, doubly clamped, divided and ligated. The splenic vein in this area is then dissected circumferentially from surrounding tissue and a tape is placed around it. With gentle traction on the tape dissection is continued in a distal direction until a segment of 4 to 6 cm. of the vein has been freed. Care is exercised to avoid troublesome hemorrhage that occurs when tributaries are torn from the splenic vein [4] (Fig. 6). Next a transverse incision is made in the posterior peritoneum and the left renal vein is isolated as it crosses the aorta. The vein is freed of surrounding tissue in

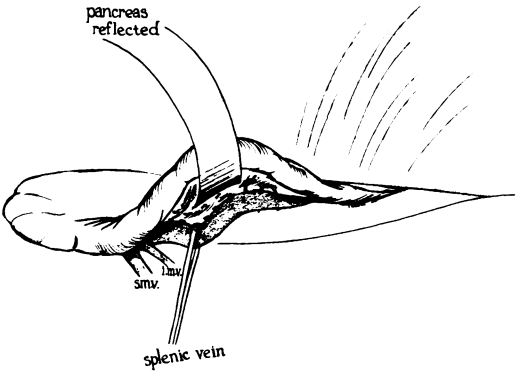


FIG. 6. The splenic vein has been identified. By careful division of pancreatic tributaries, a segment of splenic vein is isolated. The usual relationship of the splenic vein to the inferior mesenteric vein (i.m.v.) is shown. During the surgical procedure the superior mesenteric vein (s.m.v.) is not dissected and usually is not seen.

a distal direction. Cephalad to the vein the left renal artery is recognized and encircled with a tape. Pressures in the renal vein (FRV) and splenic vein (FSV) are recorded. The splenic vein is cross clamped and the pressure on the hepatic side of the vein is measured (HOSP). The pressure on the splenic side of the vein is then obtained (SOSP).

The splenic vein at its junction with the inferior mesenteric vein is doubly clamped, divided and the proximal end is oversewn. The left renal artery is temporarily occluded and a segment of renal vein is isolated between vascular clamps. An incision approximately 2.5 cm. long is made in the superior aspect of the renal vein and the distal end of the cut splenic vein is beveled appropriately. Tortion and kinking of the splenic vein are avoided and a continuous simple coaptation suture is used for the posterior wall of the anastomosis. Interrupted sutures are used for the anterior wall. The clamps on the renal vein, splenic vein and finally the renal artery are released (Fig. 7, 8).

Following completion of the distal splenorenal shunt the splenocolic and then the gastrocolic ligaments are divided. The gastrosplenic ligament and the vessels coursing through it are preserved. The greater curvature of the stomach is lifted upward, the left gastric artery is divided, and the coronary vein is identified, divided and obliterated with a continuous suture. The gastrohepatic ligament is divided (Fig. 9). Pressure in the splenic vein (FSV), renal vein (FRV) and in the superior mesenteric vein (HOSP) are again recorded.

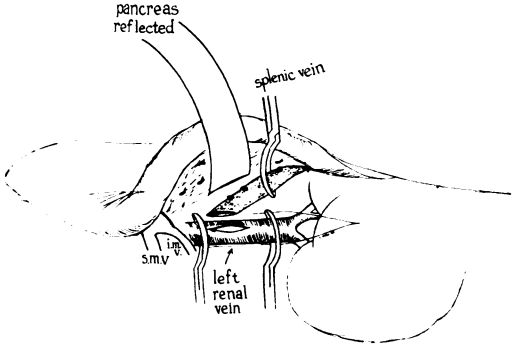


FIG. 7. The splenic vein has been divided and the distal orifice has been oversewn in a manner which leaves no cul de sac from which a thrombus could propagate. The renal vein has been isolated and incised. Before application of vascular clamps, the renal artery, not shown, is occluded. The relationship of the inferior (i.m.v.) and superior (s.m.v.) mesenteric veins is shown.

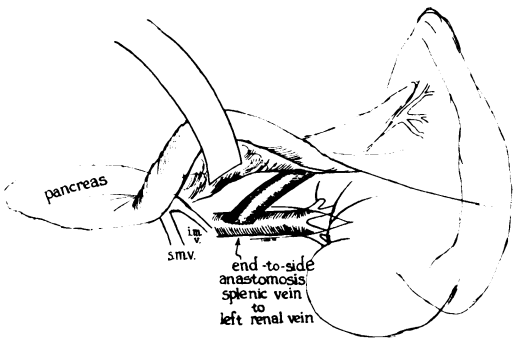


FIG. 8. Upon completion of the anastomosis the splenic vein is unkinked and without tortion even when the pancreas is allowed to return to its normal position.

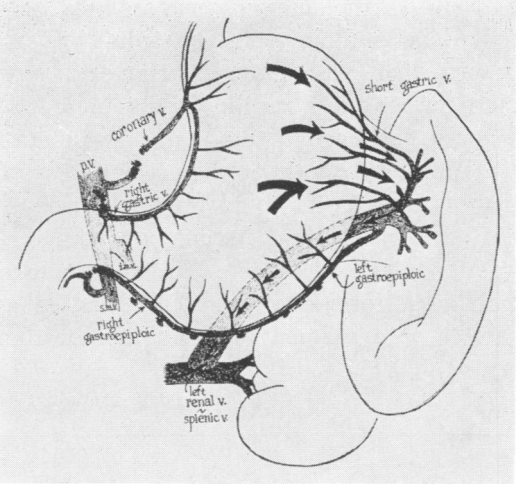


FIG. 9. The completed operation is depicted diagrammatically. Arrows indicate the direction of blood flow. The distal splenorenal anastomosis has been completed and the coronary vein has been ligated. The gastrocolic ligament has been divided and the right gastroepiploic vein ligated. At the time the gastro hepatic ligament is divided the right gastric vein is ligated. Blood from the inferior (i.m.v.) and superior (s.m.v.) mesenteric veins continues to perfuse the liver by way of the portal vein.

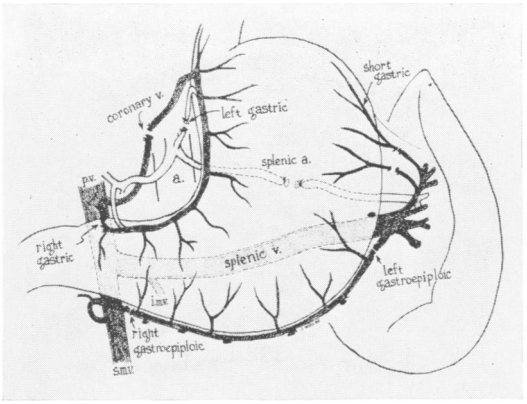


FIG. 10. The left gastroepiploic artery (not shown) and vein are ligated and the gastrocolic ligament is divided. The right gastroepiploic artery (not shown) and vein are similarly ligated as are the short gastric vessels. The coronary vein is obliterated, the left gastric artery divided near its origin and finally the splenic artery is divided.

With increasing experience the technic of the operation has been modified. Initially a long left subcostal incision was used, the pancreas was approached by freeing the splenic flexure of the colon and reflecting it caudad and in a medial direction and the splenic vein was isolated distally. The midline incision resulted in exposure that was more satisfactory; but more importantly, ascitic fluid which frequently has been a temporary postoperative problem does not dissect into tissue planes. It is easier to identify and isolate the splenic vein near its junction with the inferior mesenteric vein, the anastomosis can be performed after mobilization of a shorter vein segment and ligation of the splenic vein at its junction with the inferior mesenteric vein leaves no cul de sac in which a thrombus may form and propagate. Exposure of the pancreas through the transverse mesocolon is satisfactory and the pancreas can be mobilized, rotated and retracted with considerable less trauma to the organ.

## Technic of Gastric Devascularization with Spleen in Situ

When a distal splenorenal shunt is not performed devascularization of the greater curvature of the stomach is the first step in the procedure. The left gastroepiploic artery and vein are divided distal to the short gastric vessels and beginning at this point the gastrocolic ligament is divided close to the stomach as far as the level of the pylorus. Here the right gastroepiploic artery is ligated. The short gastric vessels are divided and the coronary vein is obliterated in the manner previously described. The left gastric artery is ligated and divided near its origin leaving the right gastric artery as the sole blood supply to the stomach. Finally the splenic artery is ligated but the spleen is left in place (Fig. 10).

## Case Reports

**Case 1.** Two years prior to admission this 59-year-old man had a cholecystectomy for chronic cholecystitis and cholelithiasis. At the time of operation there was perisplenitis and a liver biopsy was interpreted as "chronic portal hepatitis." Approximately 1 year prior to admission the patient

TABLE 1. *Laboratory Values Prior to Operation*

| Case | Hg mg./ 100 ml. | WBC | Platelet Count | Pro-thrombin Activity % | Bilirubin Total mg./ 100 ml. | Bilirubin 1 Min. mg./ 100 ml. | Alkaline Phosphatase BU | SGOT units | Total Protein mg./ 100 ml | Albumen mg./ 100 ml |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 12 | 4,600 | 90,000 | 100 | 1.40 | 0.45 | 25.6 | 52 | 5.9 | 2.72 |
| 2 | 10.6 | 2,350 | 80,000 | 100 | 1.00 | 0.22 | | 42 | 6.5 | 4.12 |
| 3 | 13.2 | 4,950 | 74,000 | 86 | 1.50 | 0.70 | 5.6 | 70 | 6.6 | 2.31 |
| 4 | 7.9 | 8,800 | 232,000 | 56 | 1.34 | 0.47 | 2.7 | 84 | 7.5 | 2.60 |
| 5 | 11.8 | 17,500 | | 70 | 1.60 | 0.64 | 3.3 | 90 | 5.5 | 2.62 |
| 6 | 11.6 | 6,300 | 130,000 | 100 | .30 | | 2.9 | 16 | 6.0 | 3.15 |

TABLE 2. *Hemodynamic Data*

| | Preoperative | | | | At Operation | | | | Postoperative | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Pre-Shunt | | Post-Shunt | | | | | |
| Case | EHBF $Au^{198}$ | CSP mm. Hg | Hepatic Venogram Category | Portogram Stage | CSFP mm. Hg* | Corrected Occluded Splenic Pressure mm. Hg | CSFP mm. Hg* | CFPP mm. Hg | EHBF $Au^{198}$ | CSP mm. Hg | Hepatic Venogram Category | Portogram Stage |
| 1 | 0.31 | 15 | 1 | 1 | | | 0 | 17 | | | | |
| 2 | 0.22 | 18 | 1 | 1 | 26 | 26 | 0 | | 0.22 | | | 3 |
| 3 | 0.21 | 22 | 2 | 1 | | | | | | | | |
| 4 | <0.12 | 15 | 2 | 1 | 19 | 27 | 4 | 8 | <0.12 | 19 | 4 | |
| 5 | 0.21 | 16 | 3 | 1 | | | | | 0.18 | 17 | 2 | |
| 6 | 0.22 | 17 | 1 | 1 | 18 | 22 | | | 0.27 | | | |

* Corrected free splenic vein pressure = free splenic pressure — renal vein pressure.

developed ascites which responded to medical therapy. Two weeks before coming to the hospital he had the first of two hematemeses necessitating transfusion with a total of 5,500 ml. of whole blood. On admission the patient appeared comfortable, the abdomen was protuberant with ascites, the liver was not palpable, but the spleen could be felt three finger breadths below the left costal margin. Palmar erythema and spider angiomata were absent. There was no peripheral edema. Liver function studies were performed (Table 1). With treatment the patient became free of ascites and upper gastrointestinal series was interpreted as being compatible with small esophageal varices. An hiatal hernia was present. Blood flows using radioactive colloidal gold were normal (Table 2). When scanned the liver was thought to be of normal size. The spleen was enlarged and isotope uptake by it was high. Hepatic vein catheterization was carried out. The corrected sinusoidal pressure was 15 mm. of mercury. When an hepatic venogram was performed there was virtually no filling of the portal venous system. Contrast material left the liver by way of the hepatic vein. Because the patient seemed to have early portal hypertension with good hepatopetal portal flow operation was not advised. Three months later the patient had a small hematemesis and tarry stools. Bleeding continued and a splenoportogram was performed. Despite the fact that the inferior mesenteric and coronary veins filled and that varices were seen there was good hepatopetal flow with a prominent liver blush on the late splenoportography films. An emergency distal splenorenal shunt was performed with ligation of the splenic artery and coronary vein. Postoperatively the patient reaccumulated ascitic fluid which became infected. In spite of drainage of multiple intra-abdominal abscesses the patient died. The shunt was patent at the time of the second operation.

**Case 2.** A 65-year-old woman had been hospitalized 11 months previously and had cirrhosis of the liver, portal hypertension, hypersplenism and diabetes mellitus. Six weeks before the present admission she was admitted to a hospital because of gastrointestinal bleeding which was thought to be from esophageal varices. On the day prior to admission she again had gastrointestinal bleeding manifested by tarry stools. The patient was alert and cooperative. Jaundice, spider angiomata, palmar erythema, ascites and peripheral edema were absent. Both the liver and spleen were

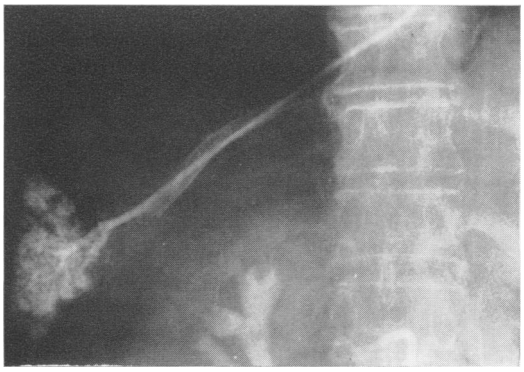

FIG. 11. Wedged hepatic venogram, Category 1. The variegated pattern in the sinusoidogram is typically present in cirrhosis. Branches of the portal vein do not fill and contrast material leaves by way of the hepatic vein.

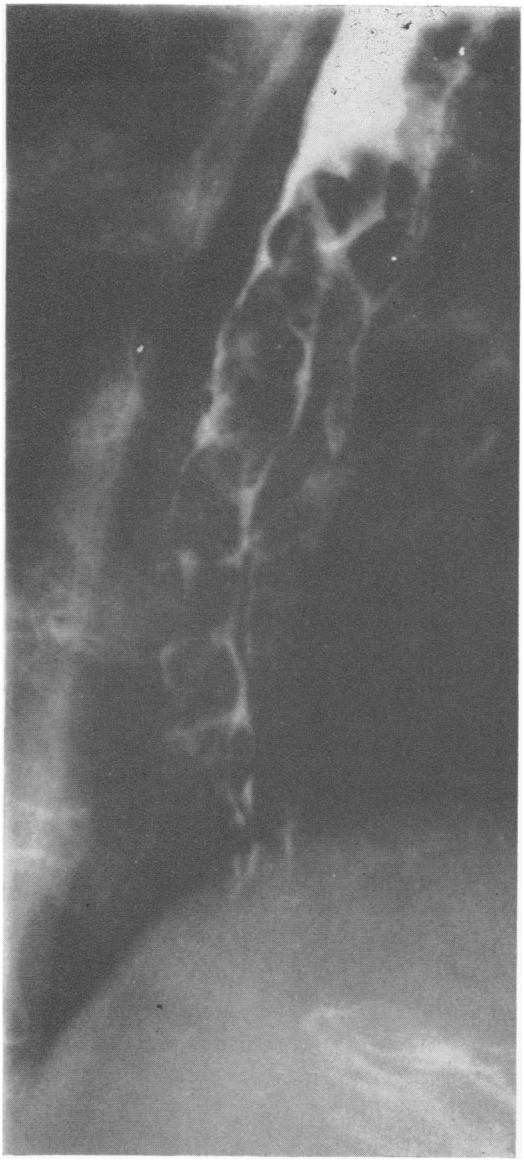

FIG. 12. Large varices are present in the lower one half of the esophagus.

moderately enlarged. Liver function tests were within normal limits but there was pancytopenia (Table 1). Estimated hepatic blood flow was K 0.22 (Table 2). Hepatic vein catheterization was carried out and the patient was found to have moderate portal hypertension. As estimated from the hepatic venogram portal flow to the liver was good (Fig. 11). On liver scan there was a high uptake of gold both in the bone marrow and in an enlarged spleen. Upper gastrointestinal series revealed large gastroesophageal varices (Fig. 12). The splenic artery was selectively catheterized and radiopaque material was injected. On the venous phase, flow was mainly towards the liver. A large coronary vein opacified to show enormous gastroesophageal collateral vessels (Fig. 13). The patient was operated upon and a distal splenorenal shunt was carried out. The coronary vein was ligated and the gastrocolic ligament was divided. Postoperatively the estimated hepatic blood flow remained unchanged. On the venous phase of the postoperative celiac arteriogram there was a large splenic vein that connected directly to the left renal vein. Blood flowed from the splenic to the renal vein and into the inferior vena cava (Fig. 14). That portal blood continued to perfuse the liver was evident from the venous phase of the superior mesentery arteriogram (Fig. 15). During the brief follow up period the patient has had no bleeding and no evidence of deterioration of liver function. Following a protein meal, ammonia levels did not rise significantly above the fasting value (Fig. 25).

**Case 3.** Two years previous to admission this 45-year-old man was seen by a physician and a tentative diagnosis of cirrhosis of the liver was made. Three weeks prior to admission the patient was admitted to another institution because of hematemesis. Medical treatment resulted in cessation of bleeding and the patient returned to full-time employment. The patient was well developed and well nourished. Spider angiomata were present over the trunk. There was no palmar erythema or gynecomastia. The liver could be palpated 2 cm. below the right costal margin. The spleen was not felt. There was no evidence of ascites or peripheral edema. Except for a platelet count of

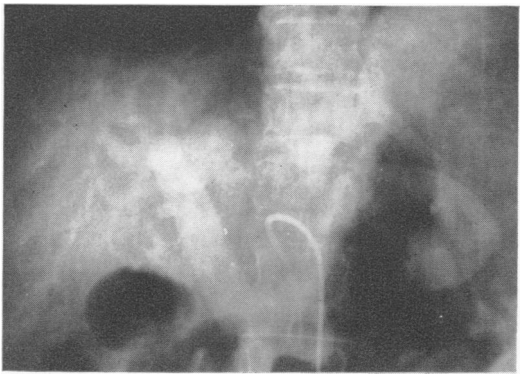

FIG. 13. Indirect portography, Stage 1. On the venous phase of the splenic artery arteriogram large gastroesophageal varices were opacified by way of the coronary vein. In spite of this portosystemic collateral hepatopetal portal flow was excellent.

74,000 laboratory findings were only mildly abnormal (Table 1). Estimated hepatic blood flows yielded values of 0.19 and 0.23 (Table 2). Hepatic vein catheterization was carried out. The corrected sinusoidal pressure was 22 mm. Hg. On the venogram contrast material left by way of the hepatic vein but a branch of the portal vein also opacified. A splenoportogram was performed and flow to the liver was only slightly decreased. The inferior mesenteric vein filled. Short gastric veins filled and formed varices in the fundus of the stomach. Other collateral veins were also present in the upper abdomen. A liver scan was interpreted as showing increased uptake of gold by the bone marrow and especially by the spleen. The liver appeared to be of normal size and the spleen was only slightly enlarged. A distal splenorenal shunt was performed. Postoperatively the patient did poorly and died in hepatic coma. At autopsy the hepatic artery and portal vein were patent but there was extensive necrosis of the liver. A laminar thrombus was present in both the splenic and renal vein but the shunt was patent.

**Case 4.** Sometime in the past, this 43-year-old Negro woman had been told that she had cirrhosis of the liver. The day prior to admission she had vomited several cupsful of bright red blood. There had been no previous hematemesis or melena. The patient was well-developed and moderately obese. She appeared comfortable. The sclera were mildly icteric, the abdomen was protuberant with no evidence of ascites and the liver and spleen were enlarged greatly. There was no palmar erythema or spider angiomata. On admission hemoglobin was 7.7 Gm./100 ml. Liver function tests were slightly abnormal (Table 1). The patient stopped bleeding spontaneously and upper gastrointestinal series showed esophageal varices. Hepatic blood flow was estimated using radioactive colloidal gold, but disappearance curves were too low for accurate calculation (Table 2). Liver scan showed a diffuse irregularity in the distribution of the isotopic material throughout the markedly enlarged liver. There was considerable uptake by an extremely large spleen as well as by the bone marrow. At the time of hepatic vein catheterization the corrected sinusoidal pressure was 15 mm. Hg. The sinusoidogram was abnormal. Although branches of the portal vein filled, venovenous shunts filled hepatic veins and it was by this route that the contrast material left the liver. A splenoportogram was performed. The coronary vein filled as did extensive collateral veins in the area of the fundus of the stomach and lower esophagus. A collateral vein, present in the right upper quadrant, was thought to be the umbilical vein (Fig. 16). Flow to the liver was good and an excellent hepatogram was obtained on the late films. A distal splenorenal shunt was carried out, the coronary vein ligated and the gastrohepatic, gastrocolic and

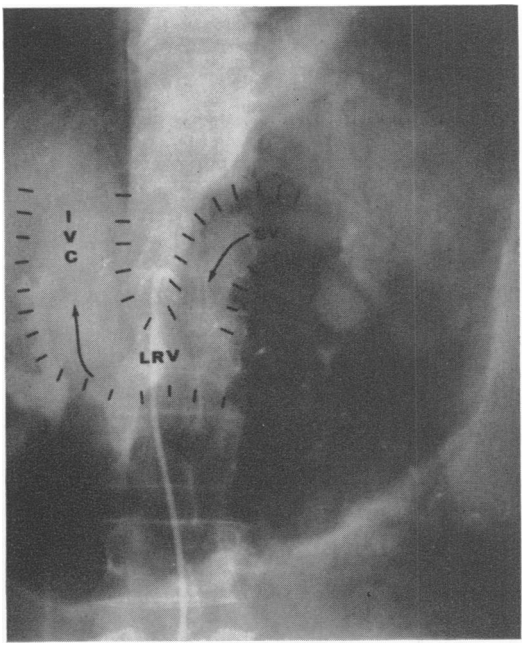


FIG. 14. Postoperative celiac angiography. On films obtained during the venous phase of a celiac axis arteriogram, contrast material filled the splenic vein (SV), entered the left renal vein (LRV) and flowed into the inferior vena cava (IVC).

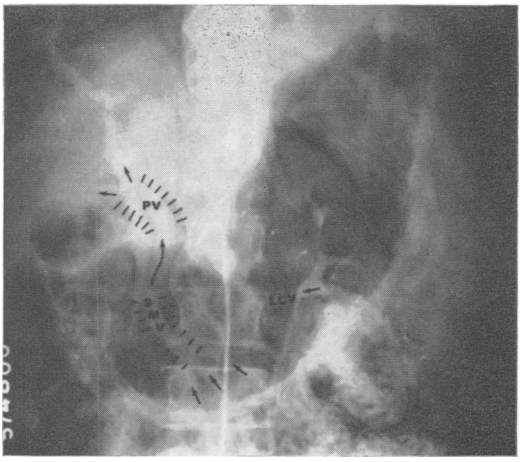


FIG. 15. Postoperative superior mesenteric angiography. A superior mesenteric arteriogram was performed. During the venous phase of the examination contrast material from the left colic vein (LCV) and superior mesenteric vein (SMV) flowed in an hepatopetal direction to fill the portal vein (PV) and its intrahepatic branches.

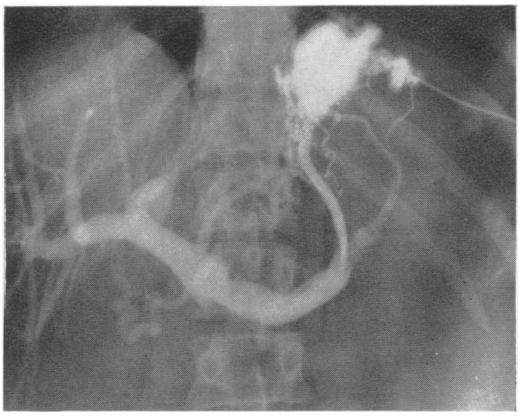

FIG. 16. Splenoportogram, Stage 1. Hepatopetal portal flow was good but contrast material filled the coronary vein and opacified varices in the area of the fundus and lower esophagus. A large collateral vein in the right upper quadrant was also opacified.

splenocolic ligaments were divided. Prior to the shunt, the renal vein pressure was 13 mm. Hg and the pressure in the splenic vein was 32 mm. Hg. The occluded splenic vein pressure was 40 mm. Hg. Post-shunt the free renal vein pressure was 12 mm. Hg and that of the free splenic vein was 16 mm. Hg. The pressure in the portal vein was 24 mm. Hg. Postoperatively the patient was again catheterized; the corrected sinusoidal pressure was 15 mm. Hg. On the hepatic venogram contrast material flowed in a retrograde direction to fill the portal vein (Fig. 17). Postoperative indirect portography was unsatisfactory but a functioning shunt was present when the splenoportogram was repeated (Fig. 18). When the gastrointestinal series was repeated there was no evidence to suggest esophageal varices.

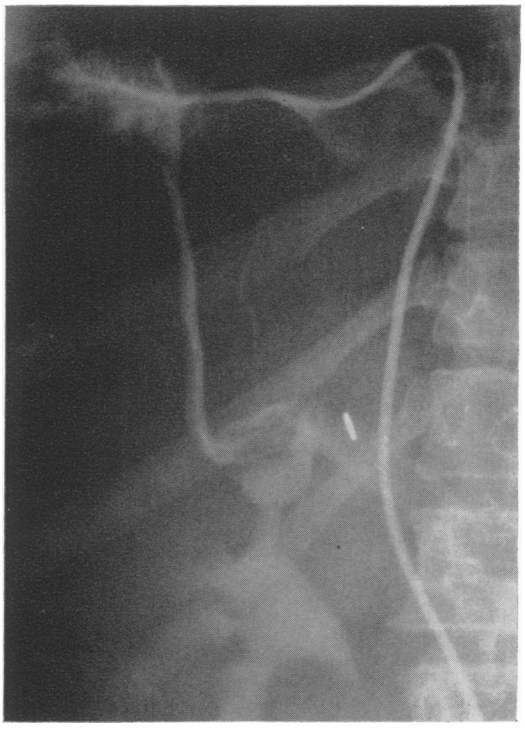

FIG. 17. Wedged hepatic venogram, Category 4. Contrast material injected with the catheter wedged in an hepatic vein drained in a retrograde direction and opacified the portal vein.

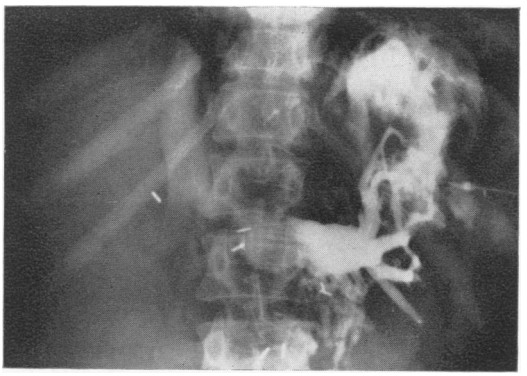

FIG. 18. Splenoportogram. Contrast material left the spleen by way of the splenic vein and flowed into the left renal vein and its tributaries and inferior vena cava. The lack of uniformity of the contrast material is the result of the Valsalva maneuver carried out by the patient.

**Case 5.** Nine months prior to admission this 40-year-old man experienced progressive increase in abdominal girth, lethargy, and weakness. Two weeks prior to admission he vomited bright red blood. There was associated melena. He received 1,000 ml. of whole blood and bleeding stopped spontaneously. An upper gastrointestinal series was said to be diagnostic of esophageal varices. One week later he had another small hematemesis but continued to have melena. He had an abdominal paracentesis and was transferred to this institution. There had been no previous history of jaundice, hepatic coma, or other difficulty. The patient appeared to be well developed and well nourished. Spider angiomata were present over the chest and shoulders, the abdomen was distended with ascites and there was edema of the abdominal wall, scrotum, and lower extremities. The liver edge reached the umbilicus but the spleen could not be palpated. The hemoglobin was low and values for total protein and albumen were decreased (Table 1). The thoracic duct and an hepatic vein were catheterized. The corrected sinusoidal pressure was

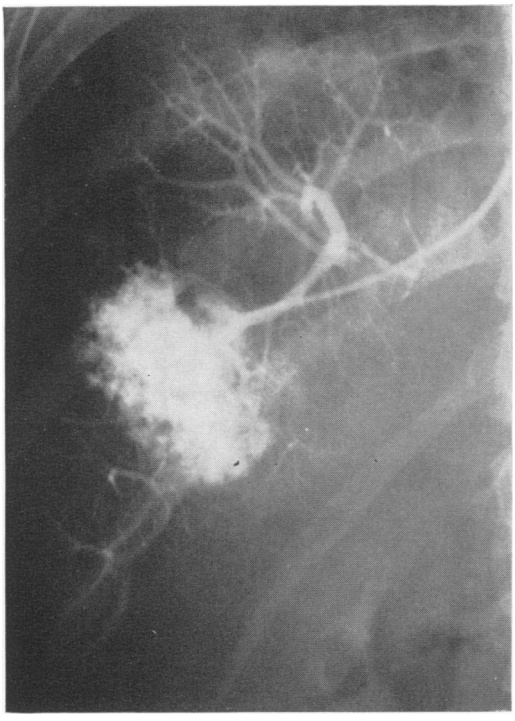

FIG. 19. Wedged hepatic venogram, Category 3. In this examination, done in the presence of tense ascites, branches of the portal vein were well filled with contrast material.

16 mm. Hg and on wedged hepatic venogram there was filling of portal vein branches (Fig. 19). With diuretics, drainage of thoracic duct lymph and replacement with albumen and plasma the patient's weight dropped from 190 to 168 pounds. Blood flows using radioactive colloidal gold yielded K values of 0.22, 0.21, and 0.20 (Table 2). The patient was discharged to be readmitted at an elective time. Two days later he returned following a large hematemesis. Splenoportogram showed good flow to the liver but there was filling of a coronary vein and a left umbilical vein. Varices were epresent in the gastroesophageal region (Fig. 20). At operation the stomach was devascularized; the spleen was left *in situ* but the splenic artery was ligated. Postoperatively the patient accumulated ascites that was controlled with diuretics. An hepatic vein catheterization done prior to discharge was similar to the preoperative examination (Fig. 21).

**Case 6.** A 69-year-old woman was told she had "an enlarged liver" 6 years previously. Shortly thereafter her spleen was also found to be enlarged. Two months prior to admission the patient had two small hematemeses for the first time.

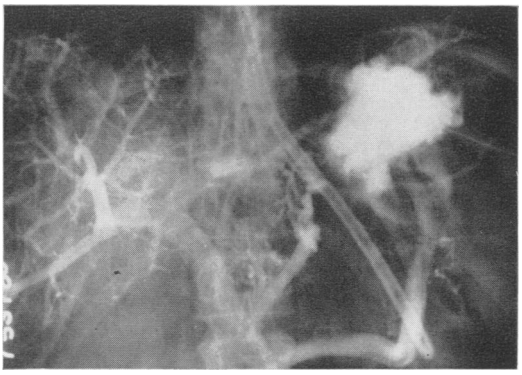

FIG. 20. Splenoportogram, Stage 1. A large coronary vein filled with contrast material which then opacified gastroesophageal varices. Portal flow to the liver remained excellent.

She was not hospitalized. Ten days before admission she had a large hematemesis and vomited "one and one-half quarts" of blood. Bleeding ceased on medical therapy. The patient had not had an illness that was suggestive of hepatitis but had experienced ascites in the past. The patient appeared well developed, well nourished and was comfortable and cooperative. The abdomen was obese but without ascites. The liver could be pal-

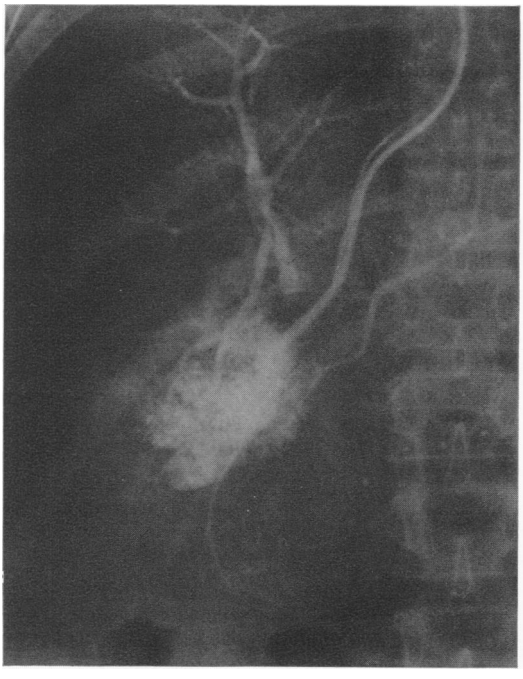

FIG. 21. Wedged hepatic venogram, Category 2. Fewer branches of the portal vein filled with contrast material and some of it can be seen leaving the liver by way of an hepatic vein.

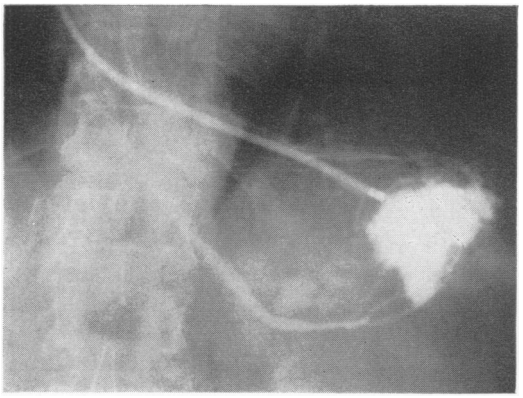

FIG. 22. Wedged hepatic venogram, Category 1. A left hepatic vein was catheterized. Although an excess of contrast material flooded the sinusoids, hepatic venovenous shunts filled and only hepatic veins were opacified.

pated three finger breadths below the right costal margin and the spleen was also readily palpable. Palmar erythema was present but there were no spider angiomata. The values obtained by various liver chemistry determinations were almost normal (Table 1). On the upper gastrointestinal series there were changes attributed to esophageal varices. Estimated hepatic blood flows using radioactive colloidal gold were carried out and yielded K values of 0.20 and 0.25 (Table 2). A liver scan was performed. The uptake of the isotope in the slightly enlarged liver was diffusely irregular. There was considerable uptake in an enlarged spleen. Hepatic vein catheterization was carried out. The corrected sinusoidal pressure was 17 mm. Hg. On the venogram there were hepatic venovenous shunts that allowed all the contrast medium to leave by way of the hepatic veins (Fig. 22). A splenoportogram was performed. The

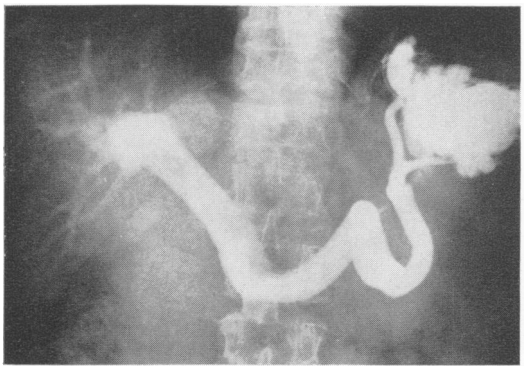

FIG. 23a. Splenoportogram, Stage 1. The coronary and short gastric veins filled and opacified varices in the gastroesophageal area.

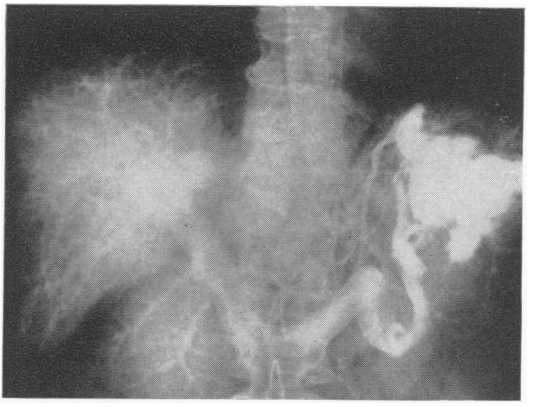

FIG. 23b. Splenoportogram, Stage 1. On late films there was a dense hepatogram, evidence of good hepatopetal portal flow.

circulation was mainly hepatopetal with a very prominent liver blush during the capillary phase. The coronary vein filled. Collaterals were present in the gastroesophageal area which were supplied by the short gastric veins as well as by the coronary vein (Fig. 23). At operation a distal splenorenal shunt was unsuccessful and the splenic artery and vein were ligated. Distal to the short gastric veins the greater curvature of the stomach was devascularized. The entire lesser curvature of the stomach was devascularized and the coronary vein was ligated. Postoperatively there has been no further bleeding.

## Discussion

Portacaval shunting remains the surest way to prevent death from bleeding esophageal varices in the patient with a patent portal vein.[7, 15] The success of the procedure, however, is markedly diminished by untoward effects upon the liver and its metabolic processes.[19] This has been emphasized by critical studies in Boston [2, 7] and Hartford [3] which have failed to reveal a prolongation of life by such procedures (Fig. 1). On the other hand, there are unquestionably many patients who tolerate a portacaval shunt with little disability and remain free of the danger of exsanguinating hemorrhage while leading an essentially normal life.[8, 26] One approach to this problem has been the attempt to identify physiologic features which can be utilized to predict the response of a given

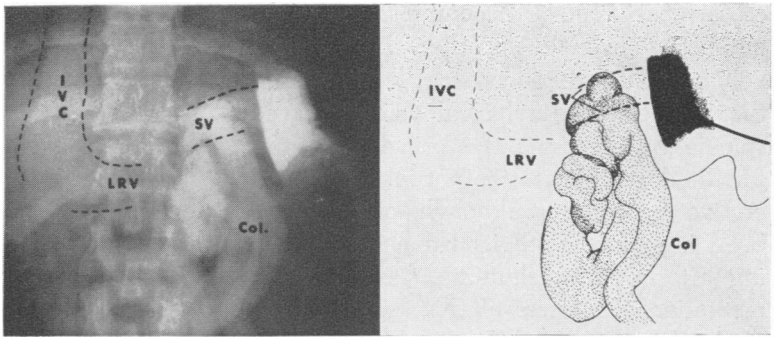


FIG. 24. Splenoportogram, Stage 4. Contrast material left the spleen by way of the splenic vein (SV) and a large caudally directed collateral (Col.). The left renal vein (LRV) and inferior vena cava (IVC) opacified by way of a spontaneous shunt. The portal vein was patent.

patient to a portacaval shunt.[26] If such data were available, a fraction of patients with bleeding esophageal varices (perhaps 40–50%) could be safely selected for the shunt procedure. Although preliminary data has indicated some of the hemodynamic changes which might account for an untoward response to the operation, the reliability of such judgment has not been established and the complexity of the disease makes an easy definition of such patients very unlikely. Even if appropriate technics of selection emerge in the near future, there remains a large group for whom a shunt procedure would likely be detrimental. The non-shunting procedures, advocated chiefly by Dr. Womack and his associates at Chapel Hill, have added considerably to the management of these patients. However, the high incidence of recurrent bleeding, both in the immediate and late postoperative periods, has clearly detracted from the achievements of this operation.[13, 16, 28] Unfortunately, non-operative management has not improved significantly and long-term survival is in constant jeopardy due to gastrointestinal hemorrhage.[7]

The major objective of this study was to develop a form of therapy which would give great protection from bleeding than non-shunting operations while avoiding the severe sequelae which may occur after sudden, complete, portal venous diversion. The concept of gastrosplenic isolation, gastric devascularization and selective venous shunting by distal splenic vein-renal vein anastomosis was developed. The rationale and specific objectives of this operation are as follows:

*1. Selective reduction of pressure and volume of flow through gastroesophageal veins.* There are isolated cases of severe bleeding from veins across an intestinal anastomosis, from hemorrhoidal veins, or simply from unknown sites in the intestinal tract. However, fatal bleeding rarely occurs from venous hypertension in areas other than the stomach and esophagus. In our series of six patients with spontaneous reversal of flow in the portal vein, the most advanced stage of portal hypertension, only one has required surgery for bleeding.[23] These patients characteristically develop extensive, caudally directed collateral, sparing the coronary-azygos system and have little in the way of esophageal varices. Indeed, the initial idea of a distal splenorenal shunt stemmed from such a patient (Fig. 24). Successful decompression of the gastroesophageal veins, however, depends upon the volume of inflow and the capacity of the outflow tract. The critical outflow systems in this procedure are the short gastric and phrenosplenic veins, the connecting link between the gastroesophageal veins and the low pressure splenic venous system. From our initial experience, there is no doubt that the splenorenal shunt completely decompresses the splenic venous system. However, if there is a large coronary vein, complete gastro-

esophageal venous decompression might not be achieved due to the relatively low volume in the venous system leading from the gastric and esophageal veins into the splenic bed. This is the principal reason for the devascularization portion of the operative procedure, although others have been impressed with its value in decreasing arteriovenous shunting.[16] Final documentation of success of this aspect of the study has not yet been accomplished. Radiographic examination by both splenic artery injection and conventional splenoportography has shown diversion of splenic flow through a patent anastomosis into the renal vein and vena cava. Splenic vein pressure has returned to normal but this does not necessarily correlate with that in the esophageal veins. Visual and tactile characteristics of the stomach have changed markedly and resemble closely those seen following a portacaval shunt. Esophageal varices have disappeared or shown a marked reduction in size by barium swallow. No patient has had variceal bleeding in the brief elapsed period since operation. The ultimate criterion for success is control of bleeding over a long period of time.

2. *Maintain portal venous perfusion of the liver.* Hepatopetal portal flow is desirable to maintain liver perfusion as near normal as possible and thereby preserve the metabolic efficiency that results from direct delivery of the products of digestion and absorption to the hepatic cells. We are firmly convinced that acute complete diversion of a large volume of portal flow often results in a serious decline in hepatocellular function. Recent studies in other laboratories have supported this concept and controlled clinical observations, discussed previously, strengthen the thesis. From the metabolic standpoint, the prominent clinical complication is portal-systemic encephalopathy. Although the precise mechanism of development is unknown, it is related to the metabolism of nitrogenous substances. Undoubtedly many other metabolic abnormalities occur following complete portacaval shunting but only a few have received more than scant attention. For example, following the ingestion of a large protein meal, the ammonia levels in portal blood are eight to ten times that of arterial blood. Therefore during a given period portal venous perfusion with 100 ml. of blood would deliver the same amount of ammonia to the liver for detoxification as would 1,000 ml. of hepatic arterial blood. Preliminary study indicates that a similar situation exists with other substances including amines, amino acids, insulin, etc. Consequently, the successful preservation of portal flow to the liver, even if small in volume, can help to insure metabolic stability.

Success in achieving this goal has been documented. On the venous phase of superior mesenteric arteriograms contrast material opacifies a patent mesenteric and portal venous system in an hepatopetal direction. The velocity of the flow appears to be decreased, a predictable finding in a system in which volume of flow is diminished while hepatic resistance remains unchanged. Hepatic blood flows using $Au^{198}$ although of limited value in this situation, have not shown a significant change. Pre- and postoperative intrahepatic pressures were essentially unchanged in Case 4.

Of equal importance has been the demonstration of the efficiency of postoperative ammonia metabolism (Fig. 25). Protein tolerance in these patients appears to be far superior to those having a portacaval shunt. Although the improvement may not be due solely to continued portal venous flow to the liver, the efficiency of substrate ($NH_3$) delivery must be an important factor.

3. *Maintain continual venous hypertension in the intestinal bed.* We have postulated that complete decompression of the portal venous system would lead to hemodynamic changes other than loss of portal flow to the liver. Prior to a portacaval shunt

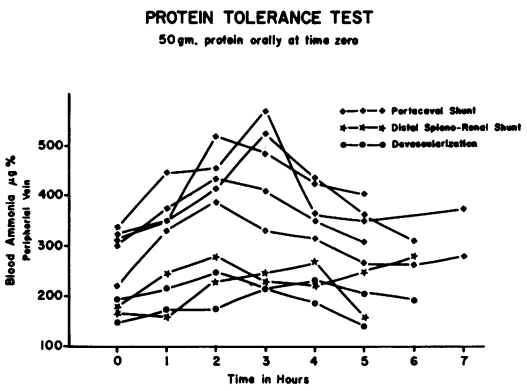


FIG. 25. Comparison in ammonia levels. Portacaval patients were admitted for study during the same time period.

splanchnic viscera have similar capillary pressures. Following the shunt these organs, except the liver, have a sudden fall in pressure to normal. The liver is the only organ with *continued outflow obstruction* and its sinusoidal pressure rarely returns to normal, frequently dropping only slightly. Under these circumstances, vasomotor control from baroreceptors should preferentially increase arterial flow to the low pressure systems. Smith *et al.*[21] have recently found evidence of this phenomenon in dogs with portal hypertension but meaningful data in the cirrhotic patient is not yet available. Another beneficial aspect of continual intestinal hypertension has recently been demonstrated by Price *et al.*[17] In both the experimental animal and man, the rate of absorption of ammonia was slowed by venous hypertension and increased following portacaval shunt. This observation may partially explain the relatively low flat protein tolerance curves of the patients having the limited shunting procedure. In Case 4 particularly, superior mesenteric angiography was interpreted as showing a very small volume of portal flow to the liver and at the time of wedged hepatic venography there was actual reversal of portal flow. In such a nearly balanced situation, the direction of flow varies depending upon volume of superior mesenteric flow or related factors. In spite of this limited flow, the protein tolerance curve was much more nearly normal than those seen following portacaval shunt.

## Clinical Application

Although it is too early to make definitive statements regarding the efficacy of the new operative approach for control of bleeding esophageal varices, some comment should be made regarding selection of patients. In our opinion the patients in whom the selective shunt or non-shunting procedure should be utilized regularly are those in whom an high volume portal flow continues to perfuse the liver. Previous studies have demonstrated the marked physiologic alterations which follow portacaval shunting.[18, 25, 26] Clinical results indicate that for any given level of hepatic integrity those patients who suffer the most severe physiologic changes are most apt to incur clinical deterioration. In addition, the far advanced, portal hypertensive has little portal flow to the liver and non-shunting or selective shunting procedures are not apt to preserve that flow.[4] This was well demonstrated in Case 4, a patient in whom a moderate amount of portal flow reached the liver preoperatively but postoperatively the portal circulation appears to be hepatopetal at one time and hepatofugal at another.

High hepatopetal portal flow and bleeding esophageal varices are most frequently encountered in the early hypertensive with significant splenomegaly. It has been documented that massive splenomegaly can cause portal hypertension and esophageal varices even in the absence of cirrhosis of the liver or of an obstructed portal vein.[27] In some patients the cirrhosis is relatively mild in terms of degree of hepatic destruction, but the increased splanchnic volume exceeds the ability of the hepatic vasculature to accommodate and portal hypertension and collateral pathways result. If the primary portosystemic collateral is the coronary vein, esophageal varices and gas-

troesophageal bleeding can occur very early in the evolution of the cirrhotic process. We believe this type of patient is almost ideally suited for the new procedure. In the same category are the so-called idiopathic portal hypertensives in whom no major obstruction to the portal venous system can be demonstrated and the liver appears to be essentially normal. The deleterious effects of portacaval shunting on such patients has been demonstrated by the excellent study of Mikkelsen *et al.* who have done much to clarify the etiology of this condition.[14]

### Non-Shunt Patients

Two patients in this series had a modification of the gastric devascularization procedure without the splenorenal shunt. In one this was done because a technical error prevented a successful anastomosis and the other was an emergent operation and a lesser procedure was decided upon. The significant variation from the technic of others is the omission of splenectomy. We have seen many large caudally directed collaterals from the spleen and destruction of such relatively harmless pathways for venous egress seems unwise (Fig. 24). Much of the morbidity and perhaps mortality of the conventional devascularization operation comes from the persistent bleeding from small vessels divided during the splenectomy. In addition to a complete "porto-azygos disconnection," the splenic artery and all gastric arteries, save for the right are ligated. This combined procedure accomplishes the immdiate control of bleeding with a lesser operative procedure. The resulting decreased pressure in collateral pathways lessens the possibility of recurrence of varices in the gastroesophageal area.

### Summary

Critical analysis of routine portacaval shunt therapy for esophageal varices has revealed a lack of success in increasing survival. Control of bleeding varices has been excellent but an increased death rate from hepatic complications has negated this achievement. Non-shunting surgical procedures have proven superior in hepatic protection, but are accompanied by a high incidence of recurrent bleeding.

A new operative procedure is described in which selective shunting is accomplished through an *in situ* spleen and a distal splenorenal shunt. The rationale of the procedure is to decompress the area critical for control of hemorrhage while maintaining a high pressure intestinal venous system and preserving portal flow to the liver.

Successful accomplishment of this goal has been demonstrated radiographically with diversion of splenic flow through a distal splenorenal shunt while superior mesenteric flow continues to perfuse the liver. The proposed metabolic advantage has been confirmed by a markedly superior response to protein tolerance testing.

From these preliminary studies, use of this type of procedure seems indicated in patients with high volume portal flow to the liver. Typical examples are seen in early cirrhosis with marked splenomegaly and in so-called idiopathic portal hypertension.

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# Book Review

**Lower Extremity Amputations for Arterial Insufficiency:** RICHARD WARREN, M.D., EUGENE E. RECORD, M.D., Boston, Little, Brown and Company, 1967, 102 pp. $12.50.

A short book on the technics of preoperative management, operation, postoperative rehabilitation, prostheses ordering, and safeguards for lower extremity amputations. Based on Warren's *Procedures in Vascular Surgery* and the authors' adopted methods; well illustrated by line drawings. Only brief mention is made of immediate prosthesis fitting; bibliography and index; attractively printed and bound; a good book for the resident's library.